CME

Rattlesnake bite in a patient with horse allergy and von Willebrand's disease: case report

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SUMMARY

Massasauga rattlesnakes are the only poisonous snakes in Ontario. While death from bites of this species is rare, the bite could cause a coagulopathy. I report a case of rattlesnake bite in a patient with asthma, horse allergy, and a documented congenital clotting abnormality.

RÉSUMÉ

Les serpents à sonnettes de l'espèce Massasauga sont les seuls serpents venimeux que l'on retrouve en Ontario. Même si les morsures mortelles sont rares, elles peuvent néanmoins provoquer une coagulopathie. Je vous présente un cas de morsure par ce crotale survenue chez un patient asthmatique, allergique au sérum de cheval et porteur d'une anomalie congénitale de coagulation.

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POISONOUS SNAKEBITES ARE rare in Canada. The only poisonous snake in Ontario is the Massasauga rattlesnake. While death from bites of this species is rare, the bite could cause, among its systemic effects, a coagulopathy. A polyvalent antivenin is available but is equine. We discuss a case of rattlesnake bite in a patient with asthma, horse allergy, and von Willebrand's disease. The current management of pit viper envenomation is also reviewed.

Case report

A 45-year-old woman presented to our emergency department with a rattlesnake bite on her left index finger. She readily identified the snake as a Massasauga rattler. This snake is not indigenous to the Toronto region but is found in the area to the northwest, around Georgian Bay.

The trapping, importation, export, and sale of exotic animals is forbidden in Ontario, but the patient admitted that she had been involved in this activity for several years, and thus was highly conversant with the complications of bites and the available antidotes and their potential complications.

Other than local pain and swelling, the patient had no symptoms. She denied having difficulty swallowing, talking, or breathing. She said she had asthma but denied any increased wheezing. She also reported that she had von Willebrand's disease and horse allergy (manifested by wheezing). History was otherwise unremarkable.

On examination, she had a resting tachycardia (pulse 118) with otherwise normal vital signs. She was anxious but in no acute distress. Her airway was clear and no adventitious breath

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Figure 1. **Patient's finger developed vesicobullous lesions the day after the bite**

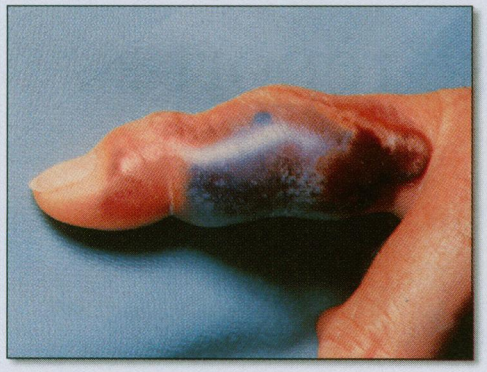
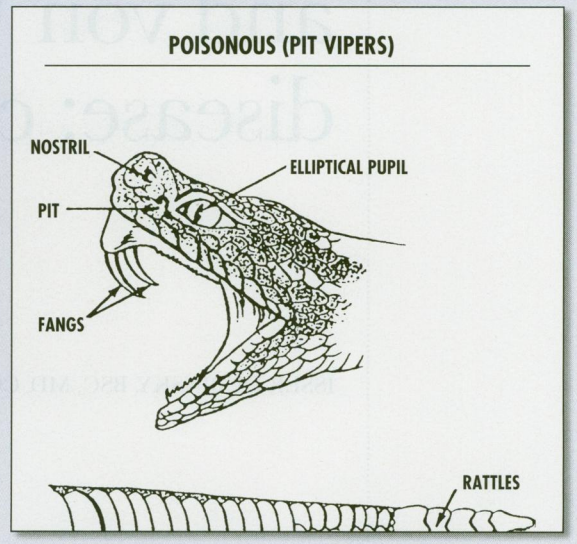


Figure 2. **Characteristics of pit vipers**



sounds were heard. Puncture wounds were noted on the ventral side of her left index finger, with considerable surrounding erythema and swelling. Movement and sensation of the hand was normal. Medical and plastic surgery services were consulted and arrangements made to have Crotalidae antivenin transported to our institution. Several hemotologic, coagulation, and biochemical parameters were measured.

Her hemoglobin level was 106, leukocyte count was 7400 with a normal differential, and platelet count was 147 000 (normal 190 000 to 400 000). Red blood cell indices were compatible with a microcytic, hypochromic anemia. Her PT was 11.0 (normal 11.0 to 14.0); INR, 1.0; activated partial thromboplastin time, 30.2 (normal 23.0 to 39.0); fibrinogen, 2.5 (normal 1.5 to 3.9); and fibrin degradation products, 10.0 (normal 0 to 9). Results of her blood sugar test, blood urea nitrogen level, creatinine level, electrolytes, liver function tests, and urinalysis were all normal. Arterial blood gases were normal as she breathed room air.

While waiting for the results of the investigations, the patient was monitored in the critical care area of our emergency department. Within 15 minutes of the bite (approximately 5 minutes after arrival), the entire palm of her hand was erythematous and edematous. Ten minutes later, the swelling extended to the wrist. After a further 45 minutes, swelling had extended to the elbow. At that time, the patient said she felt "shaky" and "lightheaded." Her vital signs remained stable.

Because the patient's symptoms were progressive, the treating physicians believed that antivenin needed to be administered. Due to relatively limited local expertise and the patient's coexisting medical conditions, we contacted experts at the Arizona Poison and Drug Information Centre in Tucson. They expressed concern about the potential hazards of administering horse-derived serum to a patient with a horse allergy and asthma. The patient was monitored continuously, and the swelling continued to progress up the arm and onto the chest wall. At no time, however, did she develop any instability of her vital signs, bronchoconstriction, or evidence of bleeding.

After repeated telephone conversations, the physicians opted to withhold the antivenin, with plans to administer it with antihistamine and

steroid coverage should the patient's clinical condition deteriorate further.

During the next day, the areas of erythema transformed into vesicobullous lesions (*Figure 1*), which broke spontaneously the following day in the intensive care unit. No other sequelae developed while she was in hospital. Her skin lesions were treated conservatively with local debridement and sterile dressings and did not become infected. She was discharged, and an appointment with a plastic surgeon was made to see whether surgical treatment of her skin lesions was possible.

Discussion

The Poison Control Centre at the Hospital for Sick Children in Toronto, Ont, had 59 reported cases of snakebites in 1990 and 28 cases between January and May 1991. Most of these bites occurred in adults, and most were by non-poisonous snakes. No further data were available from any source I contacted.

However, data from the United States (where there are considerably more poisonous snakes[6]) suggest a rate of 3.74 bites per 100 000 population. Increasingly, patients who suffer poisonous snakebites, as was the case with our patient, are engaged in illegitimate activity (approximately 50%).[12]

The most commonly encountered poisonous snake in Canada is the Massasauga rattler (*Sistrurus catenatus*) a member of the pit viper family. Pit vipers are characterized by their appearance, with a triangular head and "pits" (a heat-sensing organ) between the eyes and the nostrils. They also have elliptical pupils and two well developed fangs (*Figure 2*). They have the characteristic rattles on their tails and can grow up to 61 cm in length. They are strict carnivores.

The signs, symptoms, and severity of snake venom poisoning from pit viper bites are extremely variable. Severe tissue damage and deaths have been reported. Important variables include the species, age, and size of the biting snake; the number and location of the bites; the depth and volume of venom deposition (approximately 20% of bites do not result in any envenomation, and the volume of venom injected can range from 25% to 75% of the animal's reserve); the condition of the snake's fangs; and the duration of contact. Victim factors, such as age, general health, and quality and timing of first aid will also determine outcome.

The degree of envenomation is always difficult to determine. North American pit vipers tend to deposit their venom superficially, but deep deposition of venom could result in systemic manifestations with a relative lack of local findings. Pit viper venom contains proteolytic enzymes that alter small vessel permeability, allowing leakage of plasma and blood into tissues. Some species will also produce venom containing enzymes that alter coagulation. While some pit vipers (eg, the Mojave rattler) also produce a neurotoxin, it is of no clinical significance.[9]

The manifestations of pit viper bites are best categorized as local and systemic (*Table 1*). Systemic reactions can be classified as minor or severe. Most bites (more than 90%) are on the extremities, and reactions appear over 8 hours. If vesicles or hemorrhagic blebs appear, they generally do so (as occurred in our patient) within 24 hours.

Bleeding abnormalities seen in severe systemic reactions can result in bleeding from any site. If death occurs, it is frequently due to destruction of erythrocytes and changes in pulmonary capillary permeability leading to pulmonary edema and hemoconcentration.

Treatment

Whenever possible, treatment should begin immediately. Hikers, campers, and others involved in outdoor activity should engage in preventive measures, including wearing high-cut leather boots, lifting rocks and logs before reaching under them, and using flashlights at night.

Controversy exists about field management of bites. Snakebites by poisonous species, and particularly the Massasauga rattler, are rarely fatal. Even bites by more lethal species will be lethal within 1 hour in only 4% to 5% of cases, and within 6 hours in approximately 17% of cases.[8] Thus, the

Table 1. **Manifestations of pit viper bites**

LOCAL MANIFESTATIONS
Puncture wound
Swelling
Vesiculation (could be hemorrhagic)
Necrosis
Pain
MINOR SYSTEMIC MANIFESTATIONS
Weakness
Faintness
Nausea
Sweating
Numbness, paresthesia
Fasciculations
SEVERE SYSTEMIC MANIFESTATIONS
Hypotension
Abnormal bleeding and clotting
Hematuria
Proteinuria
Vomiting (hematemesis)
Pulmonary edema
Renal failure

need for emergency measures in the field as a lifesaving modality is less than previously thought.

The bitten area should be immobilized at or below the level of the heart and any constricting jewelry removed. The use of ice is contraindicated.[8,9] The use of incision and suction and tourniquets is controversial.[1-9,12] If a tourniquet is used, it should be applied as a broad band, proximal to the bite, and at a pressure that exceeds venous pressures but does *not* interfere with arterial flow. The tourniquet should be released every 10 to 15 minutes for 90 seconds.[11] Incision and suction and tourniquets should be used only by trained professionals. Bites should *not* be excised.

Before administering antivenin, an effort to gauge the severity of the envenomation should be made. In general, bites by Massasauga rattlers are among the least severe. Various authors[1-5,7,8,10] have devised specific grading systems for bite severity. These schemes are designed to grade severity according to combinations of local findings; systemic manifestations; laboratory abnormalities; and bite characteristics, such as the number of bites, time of first aid, and species of snake.

However, all bites should be treated as medical emergencies until observation and serial laboratory investigations reveal that no or minimal envenomation has occurred. All patients should have their vital signs frequently monitored and laboratory determinations (cross and type complete blood count, INR, clot retraction, bleeding and coagulation times, blood urea nitrogen and electrolytes), urinalysis, and fluid balance performed until it is clear that no serious envenomation has occurred. If a major envenomation is suspected, monitoring should continue for 4 to 5 days.

Von Willebrand's disease is a genetic disorder of coagulation, with an incidence of 5 to 10 per million. It is inherited as an autosomal dominant trait, with reduced penetrance. Rarely, it is inherited as an X-linked phenomenon. Its main clinical feature is abnormal platelet function, caused not by the platelet number, release, morphology, or aggregation, but by abnormal platelet adhesion. The von Willebrand factor is a factor VIII component that permits platelet adhesion and is deficient in patients with the disease.

Bleeding that results from the disease is usually mucosal and cutaneous, often with menorrhagia and gastrointestinal tract bleeding. Hemarthroses are rare. Bleeding time is the most easily performed diagnostic test. If replacement is necessary, factor VIII is the treatment of choice.

In this patient, where a snakebite-induced coagulopathy was a potential risk, the need to monitor bleeding time, in particular, was even more pressing. If a patient with von Willebrand's disease is bitten by a Massasauga rattler, a vigilant effort should be made to ascertain the patient's

baseline bleeding time, which should be closely monitored (every 4 to 6 hours) during the acute postbite phase.

Oxygen, epinephrine, injectable antihistamines, corticosteroids, and antivenin must be immediately available. Two intravenous lines should be established in all patients. In preparation for administering the antivenin, a skin test or conjunctival test for horse-serum sensitivity should be carried out in all patients. Details of skin testing are outlined in an insert that accompanies the antivenin.

In patients with a positive skin test, the risk of allergic reaction must be weighed against the risk of death from the bite (judged to be low in our patient).[8] We chose not to skin test our patient owing to her documented history of asthma and her certainty about her allergy to horses. Some argue that, with appropriate care in the intensive care unit, moderate rattlesnake envenomations should be treated with antivenin, even in allergic individuals.[8]

Administration of the antivenin should begin as soon as possible after the bite. The intravenous route is always preferred[1] and, though antivenin is of questionable value after 12 hours, it should be given up to 24 hours after a severe bite. The dose of antivenin varies up to 100 mL or more, depending on the severity of the bite. The initial 5 to 10 mL should be infused over approximately 5 minutes with the patient observed closely for adverse reactions. If none occur, the remainder can be infused at a maximal rate. If new or recurrent symptoms appear, an additional dose of 10 to 50 mL can be given. Symptoms of serum sickness (malaise, fever, arthralgia, swollen joints, urticaria) will often develop during the subsequent 24 days, especially if a total dose of more than 70 mL of antivenin has been injected.

Corticosteroids, other than for allergic reactions to horse serum, are of no proven value in snakebites. Tetanus, immunization, or booster doses should be given in accordance with the patient's immunization history. Soft tissue infections can be minimized by vigorous wound irrigation. If they occur, antibiotic therapy should be commenced, but antibiotic prophylaxis is unnecessary. Analgesics should be administered as needed. Electric shock therapy is of no benefit.[12] ■

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